

Leukaemia Section

Short Communication

t(7;14)(q34;q11) TRA/TRB

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Published in Atlas Database: November 2017

Online updated version : <http://AtlasGeneticsOncology.org/Anomalies/t0714q34q11ID3284.html>

Printable original version : <http://documents.irevues.inist.fr/bitstream/handle/2042/69823/11-2017-t0714q34q11ID3284.pdf>

DOI: 10.4267/2042/69823

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Abstract

Translocations and inversions involving the TRA/TRD locus at 14q11.2 and the TRB locus at 7q34 are characteristic anomalies in T-cell acute leukemia/lymphoma. In most instances, juxtaposing promoter and enhancer elements of TCR genes in the proximity of a partner gene results in their dysregulation that may be a critical step toward development of T-cell malignancies.

Keywords

T-cell lymphoblastic leukemia/lymphoma, t(7;14)(q34-35;q11), T- cell receptor genes.



Figure 1. Partial karyotypes with t(7;14)(q34;q11).

	Sex/Age	Diagnosis	Karyotype/td>
1	M/14	T-ALL	46,XY,t(7;14)(q35;q11)
2	F/18	T-ALL	46,XX,t(7;14)(q34;q11)
3	30/M	T-ALL	46-47,XY,+der(14)t(7;14)(q34;q11),+mar1 TRA-TRD/TRB
4	F/5	T-ALL	45,X,?der(X),t(7;14)(q34;q11),-9,-9,del(11)(p11),del(12)(p?13),+mar TRA-TRD/TRB
5	F	AITL	46,XX,dup(7)(q11q35),t(7;14)(q35;q11),t(7;7)(p13;q35) LN
6	F	AITL	46,XX,dup(7)(q11q35),t(7;14)(q35;q11),t(7;14)(p13;q11),add(14)(q11) LN
7	F	AITL	48,XX,+3,+7,t(7;14)(q35;q11),t(7;14)(p13;q11),t(14;14)(q11;q32) LN

Table 1. Clinical and genetic testing findings in reported patients with t(7;14)(q34-36;q11).

1. Kaneko et al., 1988; 2. Secker-Walker et al., 1992; 3-4. Cauwelier et al., 2006; 5-7. Cosimi et al., 1990. **Abbreviations:** M, male; F, female; AITL, Angioimmunoblastic T-cell lymphoma; LN, lymph node; T-ALL, T-cell Acute lymphoblastic leukemia.

Clinics and pathology

As translocation breakpoints near terminal regions of chromosomes are difficult to ascertain, included are cases with 7q35 breakpoints, probably involving TRB.

Disease

T-cell acute lymphoblastic leukemia (T-ALL) and angioimmunoblastic T-cell lymphoma (AITL).

Etiology

T-cell malignancies: 4 patients with T-cell acute lymphoblastic leukemia (Kaneko et al., 1988; Secker-Walker et al., 1992; Cauwelier et al., 2006) and 3 with angioimmunoblastic T-cell lymphoma (AITL) (Cosimi et al., 1990) (Table 1.).

Epidemiology

2 female and 1 male patients aged 5, 18 and 30 years old (Secker-Walker et al., 1992; Cauwelier et al., 2006). In addition 1 male (Kaneko et al., 1988) and 3 female patients (Cosimi et al., 1990) with 7q35 breakpoints were reported.

Prognosis

Unknown due to the rarity of reported patients.

Cytogenetics

Cytogenetics morphological

Because in translocations with breakpoints in near terminal regions of chromosomes precise breakpoints are often difficult to define, cytogenetic appearance of t(7;14)(q34;q11) may resemble t(7;14)(q35;q11) and rearrangements of TRB at 7q34 may be detected only by using FISH with appropriate probes.

Sole anomaly in 2 (Kaneko et al., 1988; Secker-Walker et al. 1992), complex karyotype in 1 (Cauwelier et al., 2006) and found as +der(14)t(7;14)(q34;q11),+mar1 in 1 T-ALL patient (Cauwelier et al., 2006). Found in association with complex anomalies including dup(7)(q11q35),t(7;7)(p13;q35),t(7;14)(p13;q11) and t(14;14)(q11;q32) in AITL patients (Cosimi et al., 1990).

Genes involved and proteins

TRB (T-cell receptor beta locus)

Location

7q34

Note

Various genes are activated in T-cell malignancies as a consequence of chromosome translocations, inversions and cryptic chromosomal

rearrangements involving sequences from the TRB locus such as: t(6;7)(q27;q34) TRB/ MYB, inv(7)(p15q34)/t(7;7)(p15;q34) TRB/ HOXA10, t(7;9)(q34;q34) TRB/ NOTCH1, t(7;10)(q34;q24) TRB/ HOXA11, t(7;11)(q34;p15) TRB/ LMO1, t(7;11)(q34;p13) TRB/ LMO2, t(7;14)(q34;q32.1) TRB/ TCL1A.

TRA- TRD

Location

14q11.2

Note

TRA/TRD is a target of chromosomal translocations and inversions including: inv(14)(q11q32.1)/t(14;14)(q11;q32.1) TRA-TRD/TCL1A, t(8;14)(q24.1;q11.2) MYC/TRA-TRD, t(10;14)(q24;q11.2) TLX1/TRA-TRD, t(11;14)(p15;q11.2) LMO1/TRA-TRD and t(11;14)(p13;q11.2) LMO2/TRA-TRD.

Result of the chromosomal anomaly

Fusion protein

Oncogenesis

Translocations between T-cell receptor genes and various oncogenes are a genetic hallmarks of T-cell malignancies. Chromosomal aberrations affecting TCR gene loci mainly involve TRA/TRD at 14q11.2 and TRB at 7q34 and affect various known oncogenes that most often code for transcription factors or proteins involved in transcriptional complexes, the deregulated expression of which is putatively implicated in T-cell oncogenesis.

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This article should be referenced as such:

Zamecnikova A. t(7;14)(q34;q11) TRA/TRB. *Atlas Genet Cytogenet Oncol Haematol*. 2019; 23(2):34-35.